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# The importance of new comprehension on the epidemiology and development of periodontal disease for effective preventive programs

# A importância de um novo entendimento sobre a epidemiologia e o desenvolvimento da doença periodontal para programas preventivos eficazes

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## ABCTRACT

This paper is a review of the literature as to relevant aspects of the periodontal disease, focusing on the need of new interpretations of its epidemiology and development, so that high risk groups acan be identified and really effective preventive programs can be developed.

**Keywords**: Periodontal disease, epidemiology, development, risk groups, prevention.

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**RESUMO**
Este artigo é uma revisão da literatura sobre aspectos relevantes da doença periodontal, enfocando a necessidade de novas interpretações de sua epidemiologia e desenvolvimento, para que grupos de alto risco possam ser identificados e programas preventivos realmente eficazes possam ser desenvolvidos.

**Palavras-chave**: Doença periodontal, epidemiologia, desenvolvimento, grupos de risco, prevenção.

## 1 INTRODUCTION

Periodontal disease still lacks new interpretations and a more modern understanding of its evolution and prevalence, so that prophylactic programs and treatments can be effectively instituted.

For a long time, it was believed that inflammatory periodontal disease was a single and isolated entity, mainly caused by plaque accumulation, and initially manifested by marginal gingivitis that, due to lack of proper oral hygiene, was established and evolved to destructive periodontitis, promoting continuous bone loss and subsequent tooth loss. More recently, it is understood that there is no simple cause and effect relationship between supragingival plaque volume and destructive periodontitis. It is also known that gingivitis does not necessarily progress to periodontitis.[1, 2,4,12,14].

Periodontal disease may manifest in several forms, presenting different clinical signs and evolution, which reflects relevant differences in the etiological factors and host susceptibility. Periodontal disease activity is believed to be cyclical in many cases, with periods of activity of various intensities followed by periods of stagnation or remission.[33]

Clinical and laboratory markers have been attempted to observe the individual's local resistance factor, which is another important component for understanding periodontal disease. Gingivitis or plaque index, pocket depth and insertion level would be, in this sense, clinical markers. Among laboratory markers, microflora present in healthy places has been studied. Gingival sulcular fluid, saliva and peripheral blood have been studied in diseased places.[4,9,13,24]

Thus, in order to be able to develop effective preventive programs against periodontal disease, it is necessary to establish which groups are at risk. Therefore, it should be relevant that the types of periodontal disease are well differentiated, and that the conditions under which the sites would develop the disease, remain healthy or return to balance.

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Given the lack of epidemiological data on the current oral health situation in Brazil, the objective of this study was the epidemiological survey of periodontal diseases with repercussion in prevention programs

## 2 LITERATURE REVIEW

The worldwide dental literature is extensive and provides important information so that basic concepts of periodontal disease can be reevaluated, which will lead to a more precise definition of risk groups, favoring more effective programs and prophylactic treatments.

Loe et al.[18] and Lindhe et al.[15] attributed the progression of periodontal disease to an increase in the number of bacteria without, however, indicating any association of a microorganism with disease onset and progression.

Loesche[22] called the "non-specific plaque hypothesis" the conception that the increase in bacterial biomass would be related to the establishment and evolution of periodontal disease.

Loe et al.[19,20,21] studied for 15 years tea plantation workers in Sri Lanka who did not practice any form of oral hygiene and who had a high rate of plaque, stone and gingivitis. In this group of individuals, three subgroups were identified based on interproximal insertion loss and tooth loss index, namely:

- Subjects with rapid progression of periodont: Subjects with rapid progression of periodontal disease (8%) - In this subgroup, the mean insertion loss was 9mm at 35 years and 12mm at 45 years, with an annual rate of destruction ranging between 0.1mm and 1mm. Tooth loss in this subgroup occurred at age 20, with progressive increase over age, with total tooth loss at age 45.

- Individuals without progression (11%): These individuals had a mean insertion loss of less than 1mm at 35 years, with an annual destruction rate between 0.005mm and 0.09mm. There was no essential tooth loss in this subgroup.

Goodson et al.[6] studied 22 patients with untreated periodontal pockets by measuring the insertion level in two locations of each tooth, measured monthly over a period of one year. There were no significant changes in 8% of the sites. In 7% of the sites, these became deeper and 11.5% became shallower. Where depth increased, this deepening was cyclic, followed by spontaneous overcoating and returns to original depth in half of the observations. Most individuals had sites that became deeper and others that became shallow in the same mouth. In six individuals, there was no case of site deepening, but 11.3% of the

observed sites became shallower. Thus, the authors suggested that periodontal disease could be characterized by a dynamic condition of exacerbation and remission and by periods of maturity.

Socranski et al.[27] described a random model in which activity surges occurred in short periods of time in specific locations. In this study, they observed sites with brief activity and immediate return to the remission period. Other sites were free of periodontal disease. Of sites with destructive periodontal activity, some had one or more late outbreaks; others did not show any further activity. The authors noted that outbreaks occurred with high frequency during certain periods of an individual's life.

According to Theilade[30], the hypothesis of specific plaque, according to which there would be a correct microbial diagnosis for periodontal disease, does not yet explain how disease progression can occur in the absence of pathogens considered of paramount importance.

Offenbacher et al.[25] analyzed data from each site for 18 months, classifying such sites as active and inactive. Patients in which at least one site had been classified as active had significantly higher levels of prostaglandin E2 in the gingival sulcular fluid than previous visits and significantly higher than in patients in which no site was classified as active. They noted that the increase in prostaglandin E2 levels preceded the onset of local activity, concluding that the measurement of these levels could determine remission status and susceptibility to insertion loss.

Studying populations from Tanzania (1986) and Kenya (1988), BAELUM et al.[1,2] observed that each tooth showed a different variation in the severity of periodontal disease, despite the high rates of plaque, stone and gingivitis. They also showed that periodontitis can not be considered as a consequence of gingivitis, necessarily leading to tooth loss. According to the authors, there are different levels of susceptibility. They concluded that generalized destructive periodontitis is not common in many populations, although oral hygiene is poor, there is severe gingivitis and limited availability of dental care.

Haffajee & Socransky11 (1986) attempted to determine the relationship between past periodontal destruction pattern and clinical and microbiological parameters. To this end, 61 individuals with periodontal disease aged 12 to 61 years were studied. Six sites per tooth were evaluated based on clinical indicators such as erythema, plaque, suppuration, bleeding on probing, pocket depth and insertion level. Regarding the pattern of insertion loss, these individuals were divided into three distinct groups:

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- In Group 1 in 66% of the sites there was no insertion loss. Insertion loss occurred at sites separately. Localized vertical bone loss suggestive of juvenile periodontitis was the radiographic finding of these individuals.
- In Group 2, more than one third of the sites had moderate or advanced bone loss, and the radiographic findings of these individuals revealed deep infraosseous pockets near apparently normal sites.
- Finally, there was a uniform pattern of insertion loss in Group 3, with few or no normal sites. Generalized horizontal bone loss was the radiographic finding of this group.

Regarding microbiological parameters, the most important difference was the increase in the proportion of *E. corrodens*, *S. intermedius* and *F. nucleatum* in Groups 2 and 3, which responded worse to therapy.

Genco[5] suggested that periodontal disease could be induced by both quantitative and qualitative microflora changes, which would modify its balance with the host, allowing for the establishment of an inflammatory response that could be considered an opportunistic infection, which would since have received increasing attention.

By analyzing the insertion level distribution, Cohen et al.[3] suggested three loss patterns: at least one third of all sites; more widespread disease with multiple peaks and insertion loss normally distributed in all affected sites. The findings of this study confirmed the hypothesis that different disease processes are associated with different patterns of insertion loss.

Gunsolley et al.[10] evaluated the pattern of insertion loss in 47 individuals with juvenile periodontitis and 52 with generalized periodontitis, using antibodies reactive to 25 types of bacteria suspected of being pathogens of periodontal disease. The authors concluded that the hypothesis that the inability to produce a substantial antibody response to these microorganisms would be the factor that would lead to more diffuse and significant periodontal destruction.

Wilton et al.[31] reported that there were a small number of conditions that would predispose individuals to severely accelerate periodontal destruction, for example defects in neutrophil and monocyte chemotaxis. Other conditions could accelerate the progression of periodontal disease, as well as increase the damage caused by it, such as AIDS and uncontrolled insulin-dependent diabetes.

In a review study of the epidemiological evidence of the existence of high-risk groups, Johnson et al.[13] reported that the worldwide prevalence of severe periodontitis is in the range

of 7% to 15% of the adult population and suggested the need for classification of different types of gingivitis and periodontitis, in order to properly identify the high-risk groups.

Macfarlane et al.[23] studied 11 individuals with advanced chronic periodontal disease, collecting subgingival plaque from them during nine visits, in 148 pre-selected sites and 117 control sites. Regarding microbiological changes in the studied sites, no significant differences were observed in them.

Okamoto et al.[26] studied 319 Japanese individuals aged 20 to 79 years old to examine the pattern of periodontal destruction. The clinical parameters evaluated by the authors were plaque, gingivitis, pocket depth and insertion level. Results indicated poor oral hygiene, high frequency of gingivitis and loss of insertion increasing with age. In the age group between 20 and 59 years, the majority of individuals presented very little evidence of destructive periodontal disease, being more common after 60 years.

Wilton et al.[32] stated that bacteria and their products, as well as salivary factors, were not an efficient parameter for identifying risk groups. According to the authors, saliva could be useful as a source of indicators in the actual activity of the disease or as a parameter for evaluating the response to the instituted therapy.

Curtis et. al.[4] considered that gingival sulcular fluid has a wide range of molecules to be investigated because they contain potential markers derived from host, serum and subgingival plaque tissues. The authors also reported the emergence of new biochemical indicators of periodontal disease activity, among which highlighted: enzymatic activity; elevated levels of glycosaminoglycans, reflecting in the destruction of the proteoglycan component of the conjunctive tissue; and elevated levels of certain eicosanoids, possibly involved in mediating the inflammatory process.

Based on the observation of subgingival plaque activity, Maiden et al.[24] analyzed laboratory markers for periodontal disease, concluding that there is no monospecific etiology for any of the various periodontal conditions. However, they emphasized the gingivalis bacteroids (phorfiromonas) as fundamental to the understanding of the biology of periodontal disease in humans and other animals.

According to Lindhe[16] for an opportunistic infection to occur as a cause of periodontal disease, it would be necessary: the presence of a microorganism in a microbial niche; favorable environment aimed at favoring multiplication and proliferation of certain segments of microflora; and altered host resistance to render it unable to cope with the microflora present in the ecological niche.

Socransky et al.[27] concluded that, for the establishment and progression of periodontal disease to occur, the following prerequisites must be taken into account: (1) the host is susceptible; (2) the number of pathogens exceeds the defense threshold; (3) pathogen virulence; (4) beneficial bacteria should not inhibit this process.

## 3 DISCUSSION

Diagnostic techniques of periodontal disease are still inadequate, and there appears to be no perfect test for assessing disease activity.[7,16,27]

Clinical indices observed during the periodontal examination include measures based on signs and symptoms of the already established disease as well as known predisposing factors for periodontal destruction, including the degree of inflammation, the level of compromised support tissue and the level oral hygiene practiced. Evidently, the acuity, reproducibility and validity of these measures are the subject of many controversies. Therefore, the question is not without urgency: what are the signs and symptoms that best predict the prognosis of future disease and what is its importance for disease activity?

Unlike the old model of disease progression, longitudinal site monitoring of individuals with destructive periodontal disease indicated that destruction occurs in relatively short periods, followed by periods of inactivity. Subsequently, these sites may suffer further destruction, remain inactive, or simply remission of the disease may occur.[6,11,28,29]

In fact, none of the three known models of periodontal disease activity (the continuous model, the theory of causal outbreaks, and the asynchronous multiple outbreak hypothesis) is completely clear. Moreover, it is known that they are models that can interact. The application of these concepts, therefore, may help to explain clinical and radiographic alterations, but still does not lead to qualitative modifications of periodontal therapy.[8]

Socransky et al.[28] had already concluded that several factors may complicate the possibility of determining the activity of periodontal disease, being among the most important: (1) destruction may occur at random, in relation to time and location; (2) individual susceptibility among pathogens varies between patients and between sites of the same patient; (3) no single clinical evaluation can detect areas of destruction; (4) The ability of laboratory tests to predict periodontal destruction is still very limited.

The conditions of universal prevalence of periodontal disease are not the same for all members of the population. Therefore, much has been investigated to find clinical and laboratory markers for individuals and / or groups at risk. However, none of these markers in

particular was considered as a major factor in the onset of periodontal disease, always emphasizing the multifactorial etiology linked to microflora, saliva, FGC, systemic factors and other possible factors not yet researched. In this scenario, the existence of individuals at higher risk becomes a complex problem, requiring the need to perhaps create a risk assessment system, respecting the magnitude of each factor of the etiology of periodontal disease.

Fundamentally, the etiology of periodontal disease can not be credited to localized or forgotten factors. A number of variables contribute to the explanation of the indices. Individuals should be analyzed globally. Factors that are more difficult to measure, such as bacterial composition or host resistance, are mainly responsible for the evolution of periodontal disease.

Therefore, we agree with Zanata et al.[33] when recommending that, regarding periodontal disease, care and prophylaxis planning should necessarily take into account that:

- Few individuals actually have severe periodontal disease at risk of global tooth loss;
- Most individuals with periodontal disease are not at serious risk from their globally focused dentition;
- In planning to prevent destructive periodontal disease, gingivitis should not be taken into account;
- For higher-risk cases, patients should be analyzed by comparing their individual progression with population pattern;
- These higher-risk cases should also be monitored by means of assessing disease progression against insertion loss.

It is observed that there are still no reliable means of indirect assessment of insertion loss; however, all methods should be compared with the loss analysis results to obtain their sensitivity and specificity pattern. By observing these factors, care and prophylaxis programs will be comprehensive, with curative purposes and regular follow-up programs being reserved for real risk cases.

The development of this work shows us that the concepts and understanding about the etiology, activity and progression of periodontal disease have changed a lot in the last years. Thus, it is now accepted the existence of different forms of periodontal disease with different clinical presentations and progression rates, which reflects important differences in etiological factors and host susceptibility. Thus, individuals in the world population are not at the same risk for periodontal disease, but there is a minority group at high risk.

It is essential to identify this group so that effective preventive treatments can be developed. To this end, a series of researches have been conducted aiming to achieve new indices and parameters that take into account the local resistance of the host as well as other important factors: microflora, gingival sulcular fluid, saliva, peripheral blood, factors linked to immunology, biochemistry, pharmacology etc.

## 4 CONCLUSION

This review leads us to understand that periodontal disease is not widespread in the population, but is dependent on the interrelationship of complex, local and general factors. Therefore, the lack of preventive programs against periodontal disease is a direct consequence of the impossibility of correct diagnosis and prognostic evaluation performed by the professional.

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